IN THE CLAIMS

Amend the claims as follows:

Claims 1-33. (Canceled)

34. (Previously Presented) A process for propagating a mutant herpes simplex virus (HSV) comprising:

(a) a mutation in its endogenous VP16 gene wherein the mutation reduces or abolishes the ability of the protein encoded by the VP16 gene to activate viral transcription without disrupting the structural activity of the protein; and

(b) a heterologous gene;

which process comprises infecting a cell line with the mutant herpes virus and culturing the cell line,

wherein the cell line comprises a nucleic acid sequence from a non-HSV herpes virus encoding a functional equivalent of the HSV VP16 polypeptide operably linked to a control sequence permitting expression of the polypeptide in said cell line and wherein the nucleic acid sequence (i) complements the endogenous gene and (ii) does not undergo homologous recombination with the endogenous gene.

35. (Previously Presented) A process according to claim 34 wherein the functional equivalent of the HSV VP16 polypeptide is encoded by a herpes virus gene selected from a bovine herpes virus gene and an equine herpes virus gene.

36. (Previously Presented) A process according to claim 35 in which the herpes virus gene is equine virus gene is equine herpes virus 1 gene 12, or the bovine herpes virus gene BTIF.

37. (Previously Presented) A process according to claim 34 wherein the control sequence comprises a constitutively active promoter or an inducible promoter.

38. (Previously Presented) A process according to claim 34 wherein the HSV is an HSV-1 or HSV-2.

39. (Previously Presented) A process according to claim 34 wherein the mutant herpes simplex virus comprises additional mutations which functionally inactivate one or more additional endogenous genes of said virus and the cell line comprises additional nucleic acid sequences encoding functional herpes virus genes which complement said additional functionally inactive endogenous genes.

40. (Previously Presented) A process according to claim 39 wherein said additional nucleic acid sequences encode at least one of HSV-1 ICP27, HSV-1 ICP4, an equivalent of said HSV-1 ICP27 in HSV-2 or another herpes virus, and an equivalent of said HSV-1 ICP4 in HSV-2 or another herpes virus.

41. (Previously Presented) A process according to claim 40 in which at least one of said HSV-1 ICP27 or said equivalent is driven by the ICP27 promoter and said

HSV-1 ICP4 or equivalent is driven by the MMTV LTR promoter.

42. (Previously Presented) A process according to claim 40 wherein said additional nucleic acid sequences additionally encode HSV-1 ICP27 or an equivalent thereof in HSV-2 or another herpes virus.

43. (Previously Presented) A process according to claim 42 wherein the HSV-1 ICP27 or equivalent thereof is driven by the ICP27 promoter.

44. (Previously Presented) A process according to claim 34 which the heterologous gene is operably linked to a control sequence permitting expression of the heterologous gene in a mammalian cell.

45. (Previously Presented) A process according to claim 34 wherein the heterologous gene is an HSV gene that is not operably linked to the viral control sequence with which it is naturally associated.

46. (Previously Presented) A process according to claim 34 wherein the heterologous gene encodes a polypeptide of therapeutic use.

Claims 47-58. (Canceled)